Environmental Tobacco Smoke Exposure and Pulmonary Function among Adults in NHANES III: Impact on the General Population and Adults with Current Asthma

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The impact of environmental tobacco smoke (ETS) exposure on adult pulmonary function has not been clearly determined. Because adults with asthma have chronic airway inflammation, they may be a particularly susceptible group. Using data from the Third National Health and Nutrition Examination Survey (NHANES III), I examined the cross-sectional relationship between serum cotinine, a biomarker of ETS exposure, and pulmonary function among 10,581 adult nonsmokers and 440 nonsmoking adults with asthma whose cotinine and spirometry data were available. I generated residuals, which are observed minus predicted values (based on Crapo equations), for forced expiratory volume in 1 sec (FEV_1), forced vital capacity (FVC), and FEV_1/FVC ratio to adjust for age, sex, and height. In addition, I used multivariate linear regression to control for sociodemographic characteristics and previous smoking history. Most adults with and without asthma had detectable serum cotinine levels, indicating recent ETS exposure (85.7% and 83.4%, respectively). Among nonsmoking male participants, I found no evidence that ETS exposure was related to decreased pulmonary function. In the nonsmoking female stratum, the highest cotinine tertile was associated with a lower FEV_1 [–100 mL; 95% confidence interval (CI), –143 to –56 mL], FVC (–119 mL; 95% CI, –168 to –69 mL), and FEV_1/FVC ratio (–1.77%; 95% CI, –2.18% to –1.36%). Among women with asthma, the highest cotinine tertile was also associated with decreased FEV_1 (–261 mL; 95% CI, –492 to –30 mL), FVC (–291 mL; 95% CI, –601 to 20 mL), and FEV_1/FVC ratio (–1.6%; 95% CI, –3.3% to 0.19%). In conclusion, ETS exposure is associated with decreased pulmonary function in adult females, especially those with asthma. This analysis should provide further impetus for public policies that promote smoke-free environments. ***Key words*****: asthma, environmental tobacco smoke, respiratory function tests, tobacco smoke pollution.** ***Environ Health Perspect*** **110:765–770 (2002). [Online 14 June 2002]**

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Environmental tobacco smoke (ETS) exposure is widespread, affecting the majority of U.S. adults (*1–3*). A complex mixture of over 4,000 chemical compounds, ETS contains potent respiratory irritants such as sulfur dioxide, ammonia, formaldehyde, and acrolein (*1,4*). As a consequence, ETS exposure could negatively affect adult pulmonary function. Although ETS exposure during early life appears to attenuate the development of peak lung function (*1*), the impact of ongoing exposure on pulmonary function during adulthood has not been clearly determined.

Because adults with established asthma have chronic respiratory disease, they may be particularly susceptible to adverse health effects of ETS exposure. Reflecting this view, national asthma guidelines recommend that persons with asthma avoid ETS exposure (*5*). Clearly, understanding the impact of ETS exposure on health status among adult asthmatics has important clinical and public policy implications. Despite the importance of this question, existing data on the effect of ETS exposure on adults with asthma are surprisingly limited. The few previous epidemiologic studies in adults have suggested a relationship between ETS exposure and greater respiratory symptoms, medication use, and health care utilization for asthma (*6–9*). The effects of ETS exposure on pulmonary function among adults with asthma have not been well characterized.

Previous reports using data from the population-based Third National Health and Nutrition Examination Survey (NHANES III) have reported the prevalence of ETS exposure (*2,10*) and effects of ETS on childhood respiratory symptoms and pulmonary function (*11*). The present study examines the cross-sectional relationship between serum cotinine, a biomarker of ETS exposure, and pulmonary function in the general population of nonsmoking U.S. adults. The analysis also elucidated the specific impact of ETS exposure among adults with current asthma, a potentially susceptible group.

Methods

Overview. In the analysis I used data from NHANES III, which was conducted by the National Center for Health Statistics of the U.S. Centers for Disease Control and Prevention between 1988 and 1994 (*12*). In the present study, a stratified, multistage probability design was used to select a representative sample of the civilian, noninstitutionalized U.S. population. Extensive interviews, including questions on demographic and health information, were conducted with participants in their households. In specially equipped mobile examination centers or their homes, participants underwent a standardized examination, which included physical examination, pulmonary function testing, and blood sampling. In this study, I examined the cross-sectional association between ETS exposure and pulmonary function among nonsmoking U.S. adults, with a particular focus on adults with asthma. The study was approved by the National Center for Health Statistics Institutional Review Board.

Study sample. The present study included nonsmoking adult NHANES III participants ≥ 17 years of age whose serum cotinine measurements and spirometry data were available. Of the 20,050 adult participants, I excluded 4,990 persons who indicated current smoking and an additional 3,639 subjects who did not undergo spirometry, had unreliable spirometry results, or were missing serum cotinine measurements. I excluded another 840 subjects who had serum cotinine levels of ≥ 14 ng/mL, suggesting active current personal smoking (*13,14*). The present study sample includes 10,581 adult nonsmokers.

Classification of asthma. The survey interviews ascertained selected chronic health conditions, including asthma. Subjects were asked whether they had ever received a physician diagnosis of asthma: "Has a doctor ever told you that you had asthma?" Respondents who indicated an affirmative answer were then asked if they currently had the condition: "Do you still have asthma?" In this study, adults with asthma were defined as

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respondents who reported current asthma. To reduce misclassification with smoking-related chronic obstructive pulmonary disease, asthma was further defined by excluding those subjects who also reported ever having a physician diagnosis of emphysema (*15,16*).

Assessment of ETS exposure. Serum cotinine, a metabolite of nicotine, is a widely used and specific biomarker of ETS exposure (*1*). Because the half-life of cotinine ranges from 7 to 40 hr among nonsmokers exposed to ETS, cotinine reflects exposure during the previous 3–4 days (*17,18*). Using previously described methods, serum cotinine levels were measured using HPLC/atmospheric-pressure chemical ionization tandem mass spectrometry (*19*). The limit of detection for this method is 0.05 ng/mL.

Self-reported home and work ETS exposure were ascertained by the survey interview. Domestic ETS exposure was defined as an affirmative answer to the question "Does anyone who lives here smoke cigarettes in the home?" Respondents were also asked about ETS exposure at work: "At work, how many hours per day are you close enough to people who smoke so that you can smell the smoke?" Based on this question, work ETS exposure was defined as ≥ 1 hr/day.

Pulmonary function measurement. Spirometry was performed according to the 1987 American Thoracic Society recommendations (*20*). Examinees performed five to eight forced expiratory maneuvers. To classify tests for reliability, two senior quality technicians at the spirometry quality control center reviewed all tests (*19*). In the present analysis, we included only reliable test results.

Smoking. Personal cigarette smoking was ascertained using standard questions developed for the National Health Interview Survey. In this analysis, previous smoking was defined as an affirmative answer to the question "Have you smoked at least 100 cigarettes during your entire life?" and a negative answer to the question "Do you smoke cigarettes now?" (*21*). Previous research indicates that serum cotinine levels of ≥ 14 ng/mL are most consistent with personal smoking (*13,14*), so subjects who indicated no current smoking but had serum cotinine levels greater than this level were reclassified as current smokers and excluded from analysis.

Other demographic and personal characteristics. The demographic and personal characteristics that are potentially related to ETS exposure and pulmonary function were examined. I chose education as a key socioeconomic indicator, which was ascertained as the highest grade or year of school completed. Because income can be a cause or result of health status, I performed the analyses with and without controlling for income. Low family income was defined using a survey item that ascertained whether combined family income from all sources was more than or less than $20,000 during the past 12 months. The survey ascertained race/ethnicity in a standardized manner. For this analysis, I defined race/ethnicity as white, non-Hispanic, and other categories.

Statistical analysis. Statistical analysis was performed using SAS, version 6.12 (SAS Institute, Cary, NC) and SUDAAN, version 7.5 (Research Triangle Institute, Research Triangle Park, NC). In all analyses, I used sampling weights to adjust for unequal probabilities of selection and to account for nonresponse. SUDAAN was used to calculate variance estimates that account for the complex survey design (*22*).

For bivariate comparisons, I used linear regression analysis for normally distributed continuous variables and the chi-square test for categorical variables. The prevalence of ETS exposure among persons with and without asthma was evaluated using several exposure measures: any detectable serum cotinine, self-reported domestic exposure, and self-reported work exposure. Intensity of ETS exposure was based on serum cotinine level.

I determined predicted pulmonary function values using the Crapo predictive equations based on age, height, and sex (*23*). For adults with asthma, I also applied the alternative Hankinson predictive equations, which were derived from lifelong nonsmoking NHANES III participants without any reported history of asthma, other respiratory diseases, or respiratory symptoms (*24*). These predictive equations are based on age, sex, height, and race/ethnicity. Because these equations were derived from a group of NHANES III participants that substantially overlaps with the total nonsmoking group in this analysis, these equations were only applied to analyses restricted to adults with asthma. Residuals, which are observed minus predicted values, were generated for forced expiratory volume in 1 sec (FEV_1), forced vital capacity (FVC), and FEV_1/FVC ratio to adjust for age, sex, and height (and race/ethnicity, for the Hankinson equations).

The analytic aim was to determine the cross-sectional association between ETS exposure, as measured by serum cotinine level, and each pulmonary function residual. To assess this exposure–outcome relationship, I examined three groups of subjects: total group of adult nonsmoking subjects, adults who never smoked, and nonsmoking adults with asthma. Because the results appeared to differ by sex, all analyses were stratified by sex.

In each group, multivariate linear regression analysis was conducted. As a predictor variable, I evaluated serum cotinine both as tertiles (*11*) and as a continuous variable. For each pulmonary function outcome, I examined the impact of cotinine level on the residual pulmonary function measurement, which adjusts for age, sex, and height. I then

Table 1. Demographic and personal characteristics of nonsmoking adult NHANES III participants with and without asthma.

Variable	Adults with asthma	Adults without asthma	*p*-Value for difference
Sample size (unweighted)	440	10,141	—
Estimated population (weighted)	4,881,125	112,236,176	—
Age (mean, SE)	42.3 (0.91)	44.6 (0.53)	0.012
Sex (% female)	66.3	55.7	0.033
Race/ethnicity (% white)	75.6	76.0	0.87
Low income (%)[a]	30.3	27.8	0.53
Education (mean years, SE)	12.9 (0.25)	13.0 (0.12)	0.61
Previous cigarette smoking (%)	34.9	32.6	0.53
FEV_1 (mean, SE)	2.79 (0.07)	3.02 (0.03)	< 0.0001
FVC (mean, SE)	3.80 (0.10)	4.01 (0.03)	0.049
FEV_1/FVC ratio (%)	73 (0.8)	80 (0.2)	< 0.0001

Sample included all nonsmoking adults ≥ 17 years of age who had serum cotinine measurements and reliable spirometry results.
[a]Total annual family income < $20,000.

Table 2. Prevalence of exposure (95% CI) and intensity of exposure (median, 25th–75th IQR) to ETS among adult nonsmokers by asthma status.

Prevalence measure	Adults with asthma	Adults without asthma	*p*-Value
Detectable cotinine level (%)	85.7 (80.5–90.9)	83.4 (81.1–85.7)	0.38
Intensity of ETS exposure [median cotinine level (ng/mL), 25th–75th IQR]	0.19 (0.08–0.58)	0.16 (0.07–0.42)	0.92
Self-reported domestic exposure (%) (lives with a smoker)	15.8 (10.7–21.0)	14.6 (13.1–16.1)	0.63
Self-reported work exposure (%) (close enough to smell smoke ≥ 1 hr/day)	25.8 (18.8–32.8)	20.4 (18.4–22.5)	0.11

IQR, interquartile range.

performed multivariate linear regression to control for the potential confounding effects of previous smoking history, race/ethnicity, and socioeconomic status (education level and low income). Because income can be the cause or result of impaired health status, regression models with and without income were considered. Because the results were similar, only the analyses that include income are presented. I also examined previous smoking in terms of cumulative exposure (pack-years), which did not appreciably affect the results (data not presented).

To further ensure that the highest cotinine tertile was not composed of intermittent personal smokers, I repeated all analyses using a lower serum cotinine cutoff of ≥ 10 ng/mL. This additional restriction affected < 0.5% of subjects in each group: n = 50 nonsmokers, n = 26 never smokers, and n = 1 adult with asthma. I found no appreciable change in the results (data not shown).

Results

Demographic characteristics. After exclusions, the study sample included 440 nonsmoking adults with current self-reported physician-diagnosed asthma, which represents 4.9 million adults in the United States. Among the nonsmoking study sample, the prevalence of current asthma was 4.4% [95% confidence interval (CI), 3.7–5.0%]. Compared to adults without asthma, those with asthma were younger (mean age, 44.6 vs. 42.3 years), were more likely to be female (66.3% vs. 55.7%), and had lower pulmonary function measurements (Table 1). I found no statistical differences in race/ethnicity, previous personal cigarette smoking history, or socioeconomic status (educational attainment and proportion with low income).

ETS exposure. Adults with and without asthma had similar prevalence and intensity of ETS exposure (p > 0.10; Table 2). Most adults with asthma had detectable serum cotinine levels, indicating recent ETS exposure (85.7%). The prevalence of exposure was similar to those without asthma (83.4%). I also found no differences in self-reported domestic ETS exposure (i.e., lives with a smoker) or self-reported work exposure (i.e., reports being close enough to smell smoke at work ≥ 1 hr/day). Compared to adults without asthma, adults with asthma had similar ETS exposure intensity (median cotinine, 0.19 vs. 0.16 ng/mL).

Self-reported ETS exposure was related to serum cotinine level. The number of smokers at home was moderately correlated with serum cotinine (Spearman rank correlation = 0.44, p < 0.0001). Reported daily duration of workplace ETS exposure was also associated with cotinine level (Spearman r = 0.30, p < 0.0001).

ETS exposure and pulmonary function among adult nonsmokers. Among 10,581 adult nonsmokers, which represent 112.2 million U.S. adults, the association between ETS exposure and pulmonary function varied by sex. In the male stratum, the medium and high cotinine tertiles were associated with a decreased FEV_1/FVC ratio compared to the lowest tertile (Table 3). When treated as a continuous variable, higher cotinine levels were also related to decreased FEV_1/FVC ratio. In analysis adjusting for age and height, I found no statistical relationship between cotinine level and FEV_1 or FVC. After controlling for race/ethnicity, previous smoking history, and socioeconomic indicators (education level and low income), the medium cotinine tertile was associated with a greater FEV_1 and FVC compared to the lowest exposure group (mean increment, 78 and 94 mL, respectively). Despite this finding, I found no statistical association between the highest cotinine tertile and either FEV_1 or FVC.

In the female stratum, cotinine levels were negatively associated with every measure of pulmonary function (Table 3). Adjusting for age and height, the medium and highest cotinine tertiles were related to decreased FEV_1, FVC, and FEV_1/FVC ratio. In multivariate analysis controlling for additional covariates, the highest cotinine tertile was statistically associated with a lower FEV_1 (–100 mL; 95% CI, –143 to –56 mL), FVC (–119 mL; 95% CI, –168 to –69 mL), and FEV_1/FVC ratio (–1.77%; 95% CI, –2.18% to –1.36%). As a continuous variable, greater cotinine levels were also related to decreased FEV_1, FVC, and FEV_1/FVC ratio (Table 3).

When the sample was further restricted to adults who had never smoked, I observed the same pattern of results (Table 4). Among male adults, the medium and highest cotinine tertiles were associated with decreased FEV_1/FVC ratio. Although the medium cotinine tertile was related to a higher FEV_1 and FVC in the multivariate analysis, I found no statistical relation between the highest tertile and either spirometric measure. In the female stratum, the medium and highest cotinine tertiles were associated with decreased levels of every pulmonary function measure, adjusting for age and height. After controlling for the additional personal and socioeconomic covariates, the highest cotinine tertile was associated with decreased FEV_1 (–116 mL; 95% CI, –154 to –77 mL), FVC (–143 mL; CI, –187 to –99 mL), and FEV_1/FVC ratio (–1.82%; CI, –2.25% to –1.39%).

ETS exposure and pulmonary function among nonsmoking adults with asthma. Among male adults with asthma, the highest cotinine tertile was associated with a lower age- and height-adjusted FEV_1/FVC ratio, even after controlling for race/ethnicity, previous smoking history, and socioeconomic indicators (Table 5). The medium cotinine tertile was related to a higher FEV_1 and FVC. However, I found no relation between the highest cotinine tertile and either measure. As a continuous variable, cotinine level was also not related to FEV_1 or FVC.

In the female stratum, the overall pattern of results suggested a relationship between greater cotinine levels and lower pulmonary function. After controlling for all covariates, the highest cotinine tertile was associated with

Table 3. ETS exposure and pulmonary function among nonsmoking adult participants in NHANES III shown by change in mean residual value (95% CI).

	FEV_1 (mL)		FVC (mL)		FEV_1/FVC ratio (%)	
Sex/cotinine level[a]	Adjusted for age and height[b]	Multivariate[b]	Adjusted for age and height	Multivariate	Adjusted for age and height	Multivariate
Males[c]						
L	—	—	—	—	—	—
M	75 (–4 to 153)	78 (4 to 153)	86 (–6 to 179)	94 (5 to 182)	–0.57 (–0.88 to –0.26)	–0.53 (–0.80 to –0.26)
H	22 (–38 to 83)	47 (–11 to 106)	–17 (–96 to 61)	19 (–56 to 94)	–1.27 (–1.62 to –0.92)	–1.18 (–1.49 to –0.87)
Cont	–15 (–36 to 6)	–2.7 (–21 to 15)	–26 (–58 to 6)	–9 (–36 to 17)	–0.24 (–0.32 to –0.16)	–0.22 (–0.32 to –0.12)
Females[d]						
L	—	—	—	—	—	—
M	–53 (–96 to –9)	–38 (–81 to 6)	–55 (–103 to –6)	–37 (–86 to 11)	–0.64 (–1.05 to –0.33)	–0.61 (–0.96 to –0.26)
H	–142 (–189 to –95)	–100 (–143 to –56)	–169 (–221 to –117)	–119 (–168 to –69)	–1.80 (–2.21 to –1.39)	–1.77 (–2.18 to –1.36)
Cont	–46 (–60 to –33)	–38 (–51 to –25)	–51 (–66 to –37)	–41 (–55 to –27)	–0.40 (–0.56 to –0.24)	–0.42 (–0.58 to –0.26)

[a]Cotinine analyzed as tertiles: L, lowest (0–0.093 ng/mL); M, medium (> 0.093 to 3.16 ng/mL); H, highest (> 3.16 ng/mL); Cont, continuous variable. [b]Residual values calculated as observed minus expected values. Expected values derived from Crapo equations based on age, sex, and height; multivariate models also adjust for educational attainment, low income, previous smoking history (if any), and race/ethnicity. [c]No. participants = 4,491; estimated population = 49,259,167. [d]No. participants = 6,090; estimated population = 62,977,009.

decreased FEV_1 (–261 mL; 95% CI, –492 to –30 mL), FVC (–291 mL; 95% CI, –601 to 20 mL), and FEV_1/FVC ratio (–1.6%; 95% CI, –3.3% to 0.19%). The confidence interval for FVC and FEV_1/FVC did not exclude a lack of relationship. When considered as a continuous variable, greater cotinine level was statistically associated with a lower FEV_1/FVC ratio (–0.74 for each 1 ng/mL cotinine increment; 95% CI, –1.4 to –0.74).

To further evaluate the association between ETS exposure and pulmonary function among adults with asthma, I repeated the analysis using the Hankinson spirometric reference values derived from NHANES III participants with no lifetime smoking history, no asthma, and no other respiratory symptoms or conditions. I found no statistical association between cotinine level and any pulmonary function measurement among men (Table 6). In the female stratum, the highest cotinine tertile was associated with decreased FEV_1 (–250 mL; 95% CI, –487 to –14 mL), FVC (–275 mL; 95% CI, –583 to 34 mL), and FEV_1/FVC ratio (–1.9%; 95% CI, –6.1% to 2.2%).

ETS exposure at home and work. Because the pulmonary function results appeared to differ by sex, I examined the relationhip between sex and self-reported location of ETS exposure. In the entire nonsmoking sample, a similar proportion of males (14.3%) and females (14.9%) indicated home ETS exposure (p = 0.51). Nearly twice as many male subjects reported work exposure (27.7% vs. 15.2%, $p < 0.0001$). Among adults with asthma, I found also no statistical difference in self-reported home exposure between male (18.8%) and female respondents (14.3%; p = 0.41). A greater proportion of male subjects with asthma reported exposure to ETS at work (35.9% vs. 20.6%, p = 0.07).

In the male stratum of the entire nonsmoking sample and the asthma sample, the medium cotinine tertile was statistically associated with greater mean FEV_1. When I added location of exposure to these multivariate linear regression analyses, the medium cotinine tertile was less strongly associated with FEV_1 in the entire nonsmoking group (coefficient decreased by 17%) and the asthma group (coefficient decreased by 5%).

Discussion

ETS exposure was widespread among nonsmoking U.S. adults, with no evidence of selective avoidance by persons with current asthma. Among nonsmoking women, ETS exposure was associated with a moderate reduction in pulmonary function. In the subgroup of women with asthma, ETS exposure was also related to decreased pulmonary function. Compared to the general population of nonsmoking adults, the reduction in pulmonary function appeared even greater among adult women with asthma (e.g., FEV_1 decrement, –261 vs. –100 mL). In men, however, I found no evidence that ETS exposure was related to decreased pulmonary function among nonsmoking adults or those with asthma. These results support a deleterious effect of ETS on the respiratory health of women, especially those with asthma.

A significant proportion of U.S. adults report exposure to ETS, ranging from 37% to 63% (*1–3*). A previous report from NHANES III documented that most children and adults had detectable serum cotinine levels, indicating ETS exposure (*2*). Because adults with asthma have a chronic respiratory disease, they might be expected to avoid ETS exposure. Few previous studies have tested this assumption. In a cohort of adults with asthma living in northern California, 46% reported recent ETS exposure (*25*). Among adult health maintenance organization members with asthma, 38% indicated regular ETS exposure (*8*). Neither study compared subjects with asthma to a general population referent group. A population-based study from

Table 4. ETS exposure and pulmonary function among adult participants in NHANES III who never smoked shown by change in mean residual value (95% CI).

	FEV_1 (mL)		FVC (mL)		FEV_1/FVC ratio (%)	
Sex/cotinine level[a]	Adjusted for age and height[b]	Multivariate[b]	Adjusted for age and height	Multivariate	Adjusted for age and height	Multivariate
Males[c]						
L	—	—	—	—	—	—
M	114 (7 to 221)	130 (29 to 230)	134 (–8 to 277)	157 (26 to 288)	–0.49 (–0.90 to 0.078)	–0.45 (–0.84 to –0.058)
H	4 (–78 to 87)	45 (–32 to 120)	–38 (–148 to 73)	20 (–84 to 124)	–1.17 (–1.56 to –0.78)	–1.06 (–1.47 to –0.65)
Cont	–34 (–60 to –9)	–19 (–39 to 0)	–45 (–95 to 5)	–23 (–63 to 17)	–0.23 (–0.35 to –0.11)	–0.18 (–0.28 to –0.08)
Females[d]						
L	—	—	—	—	—	—
M	–43 (–84 to –1)	–24 (–66 to 18)	–56 (–111 to –0.5)	–33 (–87 to 22)	–0.70 (–1.09 to –0.31)	–0.60 (–0.97 to –0.23)
H	–168 (–209 to –126)	–116 (–154 to –77)	–209 (–256 to –162)	–143 (–187 to –99)	–2.01 (–2.42 to –1.60)	–1.82 (–2.25 to –1.39)
Cont	–58 (–78 to –37)	–44 (–63 to –25)	–65 (–88 to –42)	–48 (–69 to –27)	–0.54 (–0.71 to –0.36)	–0.51 (–0.67 to –0.35)

[a]Cotinine analyzed as tertiles: L, lowest (0–0.093 ng/mL); M, medium (> 0.093–3.16 ng/mL); H, highest (> 3.16 ng/mL); Cont, continuous variable. [b]Residual values calculated as observed minus expected values. Expected values derived from Crapo equations based on age, sex, and height; multivariate models also adjust for educational attainment, low income, previous smoking history (if any), and race/ethnicity. [c]No. participants = 2,567; estimated population = 28,631,542. [d]No. participants = 4,858; estimated population = 46,916,128.

Table 5. ETS exposure and pulmonary function among nonsmoking adults with asthma participating in NHANES III shown by change in mean residual value (95% CI).

	FEV_1 (mL)		FVC (mL)		FEV_1/FVC ratio (%)	
Sex/cotinine level[a]	Adjusted for age and height[b]	Multivariate[b]	Adjusted for age and height	Multivariate	Adjusted for age and height	Multivariate
Males[c]						
L	—	—	—	—	—	—
M	572 (91 to 1,053)	569 (78 to 1,060)	222 (–103 to 547)	222 (–92 to 536)	–0.58 (–1.9 to 0.72)	–0.54 (–1.8 to 0.73)
H	209 (–224 to 642)	242 (–169 to 653)	–84 (–424 to 256)	–30 (–331 to 271)	–1.9 (–3.1 to –0.68)	–1.6 (–2.8 to –0.30)
Cont	–85 (–197 to 28)	–74 (–162 to 14)	–42 (–167 to 84)	–24 (–107 to 59)	–0.76 (–1.1 to –0.41)	–0.74 (–0.95 to –0.53)
Females[d]						
L	—	—	—	—	—	—
M	–68 (–288 to 152)	–87 (–278 to 104)	–46 (–288 to 195)	–63 (–278 to 152)	–0.40 (–2.0 to 1.2)	–0.46 (–2.0 to 1.1)
H	–232 (–457 to –8)	–261 (–492 to –30)	–276 (–603 to 51)	–291 (–601 to 20)	–1.4 (–3.0 to 0.31)	–1.6 (–3.3 to 0.19)
Cont	–58 (–172 to 56)	–52 (–157 to 53)	–90 (–219 to 39)	–77 (–186 to 32)	–0.74 (–1.29 to –0.19)	–0.74 (–1.4 to –0.74)

[a]Cotinine analyzed as tertiles: L, lowest (0–0.093 ng/mL); M, medium (> 0.093– 3.16 ng/mL); H, highest (> 3.16 ng/mL); Cont, continuous variable. [b]Residual values calculated as observed minus expected values. Expected values derived from Crapo equations based on age, sex, and height. Multivariate models also adjust for educational attainment, low income, previous smoking history (if any), and race/ethnicity. [c]No. participants = 145; estimated population = 1,646,890. [d]No. participants = 295; estimated population = 3,234,235.

Canada found that 42% of nonsmoking children and adults with asthma reported ETS exposure during the previous 24 hr, compared with 32% of the general population (*26*). In a nationally representative sample of U.S. adults, the present analysis found no evidence that adults with asthma have a lower prevalence or intensity of ETS exposure.

In children, more than 30 studies have linked domestic ETS exposure with decreased development of lung function (*1*). Among adults, the impact of ETS on pulmonary function has been less clear. In several studies, self-reported ETS exposure was associated with decreased FEV_1, FVC, or FEV_1/FVC ratio (*27–33*). Supporting these deleterious effects of ETS, we found an increase in pulmonary function among 53 bartenders after reduction in workplace ETS exposure during an 8-week period (*34*). Two other observational studies demonstrated a relation between ETS exposure and decreased forced expiratory flow, midexpiratory phase ($FEF_{25\%-75\%}$), but not FEV_1 or FVC (*35,36*). Other investigators have found no consistent relationship between ETS exposure and pulmonary function in adults (*37–46*).

Nearly all previous studies that examined the effects of ETS exposure on adult pulmonary function have relied on self-reported exposure assessment. Ascertainment of ETS exposure by self-report is potentially subject to information bias, which limits interpretation of these studies. For example, persons with impaired pulmonary function might be more likely to remember and report ETS exposure, whereas unaffected persons might underreport ETS exposure. Alternatively, adults who develop a pulmonary function decrement from ETS exposure or other causes may subsequently avoid ETS exposure, attenuating the observed impact of ETS on pulmonary function in a cross-sectional analysis. Furthermore, nondifferential misclassification of exposure status would likely reduce the estimated effect of ETS exposure.

In one of the few other studies using objective ETS exposure assessment in adults, Carey et al. (*47*) demonstrated a cross-sectional association between greater cotinine levels and decreased FEV_1 in British adults (105-mL decrement in the highest quintile). In a smaller study of 301 Scottish adults, serum cotinine level, as a continuous variable, was not related to pulmonary function (*33*). Another small study found no impact of ETS exposure, confirmed by urinary cotinine measurement, on peak expiratory flow variability among healthy nonsmoking adults (*48*). The present population-based study, which uses a biomarker to minimize misclassification of ETS exposure, further establishes the link between ETS exposure and pulmonary function impairment.

Although adults with asthma comprise a potentially susceptible population, the impact of ETS exposure on their pulmonary function has not been well characterized. In a population-based sample of Swiss adults with asthma, self-reported workplace ETS exposure was associated with decreased FEV_1 (4.8% decrement) (*43*). Domestic ETS exposure was not examined and ETS biomarkers were not measured. Other investigators examined the impact of self-reported ETS exposure on 200 adults with asthma who were attending a university-based chest clinic in India (*7*). Compared with unexposed patients, adult asthmatics reporting ETS exposure had lower FEV_1 (68.7% vs. 80.8% of predicted) and FEV_1/FVC (63.5% vs. 78.4%). Based on data from a study in Tuscon, Arizona, there was no apparent relationship between ETS exposure and peak expiratory flow among adults with asthma (*49*). The present study provides important information that supports the relationship between ETS exposure and pulmonary function decrement among women with asthma.

Because ETS contains potent respiratory irritants, exposure may adversely affect bronchial smooth muscle tone or induce airway inflammation in adults with asthma (*1*). Controlled chamber exposure studies support the biologic plausibility of ETS-related asthma exacerbation. After short-term exposure of 1–3 hr, many asthmatics experience a decline in FEV_1 (*50–53*). Pretreatment with bronchodilators appears to prevent the acute decline in FEV_1 in previously reactive subjects (*54*). Other studies, however, have found no effect of acute chamber ETS exposure on lung function in asthmatic subjects (*55,56*). Interpretation of these controlled exposure studies is limited by small sample size, variable subject inclusion criteria, and variation in chamber exposure methodology. Nonetheless, these experimental studies support the plausibility of ETS-induced pulmonary function decline in adults with asthma.

The analysis is subject to certain limitations. Using this cross-sectional data, the analysis could not separate the acute from chronic effects of ETS exposure on pulmonary function. Previous studies have documented both acute (*57*) and chronic effects (*1*) of ETS exposure on pulmonary function. Moreover, cotinine reflects ETS exposure during the past 3–4 days. To assess the chronic impact of ETS exposure on pulmonary function requires the assumption that current exposure is a good proxy for previous exposure. Using a survey-based ETS exposure instrument, our group found that self-reported ETS exposure was stable over a 6–7 month period (*25*). Even so, the possibility of changing exposure status over time cannot be excluded.

Because this study was cross-sectional, the causal pathway cannot be clearly defined in all cases. The association between ETS exposure and decreased pulmonary function in women cannot be plausibly explained by a reverse chain of events, that impaired pulmonary function causes women to increase their ETS exposure. In men, however, the relationship between ETS exposure and higher pulmonary function in the medium cotinine tertile could be explained by selective ETS avoidance (i.e., that lower pulmonary function or associated respiratory symptoms cause men to decrease their exposure). Supporting this idea, men were more likely than women to report work exposure, a potentially more avoidable exposure location. Moreover, controlling for exposure location attenuated this relation between ETS exposure and higher pulmonary function.

Despite extensive evidence that ETS has serious long-term health consequences such

Table 6. The association between ETS exposure and pulmonary function among adults with asthma, using predicted spirometry values from NHANES III participants who never smoked and had no respiratory symptoms or conditions, shown by change in mean residual value (95% CI).[a]

Sex/cotinine level[b]	FEV_1 (mL)	FVC (mL)	FEV_1/FVC ratio
Males[c]			
L	—	—	—
M	423 (–72 to 918)	95 (–241 to 431)	7.6 (–3.2 to 18.4)
H	90 (–385 to 565)	–166 (–509 to 178)	3.7 (–6.1 to 13.5)
Cont	–47 (–131 to 37)	30 (–64 to 124)	–1.6 (–3.4 to 0.18)
Females[d]			
L	—	—	—
M	–110 (–314 to 95)	–135 (–340 to 71)	–0.5 (–4.4 to 3.4)
H	–250 (–487 to –14)	–275 (–583 to 34)	–1.9 (–6.1 to 2.2)
Cont	–44 (–163 to 75)	53 (–64 to 171)	–0.23 (–2.0 to 1.5)

[a]Residual values were calculated as observed minus expected values; expected values were derived from Hankinson equations based on age, sex, race/ethnicity (white, African American, Mexican American), and height. All results were based on multivariate models that also adjust for age, educational attainment, low income, and previous smoking history (if any). The estimated population excludes 17 subjects who did not report race/ethnicity as white, African American or black, or Mexican American. [b]Cotinine analyzed as tertiles: L, lowest (0–0.109 ng/mL); M, medium (> 0.109–3.68 ng/mL); H, highest (> 3.68 ng/mL); Cont, continuous variable. [c]No. participants = 141; estimated population = 1,565,460. [d]No. participants = 282; estimated population = 2,971,841.

as lung cancer and cardiovascular disease, exposure remains widespread. Persons who actively smoke may resist efforts to curtail their behavior. Many members of the public also do not believe that ETS poses a threat to their health. Among bartenders working in San Francisco, California, our group found that 21% expressed the belief that ETS exposure has no adverse effect on their personal health (*34*). Business owners, especially restaurant, bar, and hotel proprietors, are concerned that public smoking prohibition may result in economic losses. Taken together, these factors constitute a significant barrier to establishing smoke-free public places. The observed association between ETS exposure and pulmonary function decrement should provide further impetus for public policies aimed at creating smoke-free environments.

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